Hydrothorax is an uncommon but well-recognized complication of peritoneal dialysis. Here, we describe a case of hydrothorax in a peritoneal dialysis patient who underwent video-assisted thorascopic surgery and talc pleurodesis for a diaphragmatic defect to preserve peritoneal dialysis capabilities. We reviewed the literature for 2001 – 2005 to assess how others have handled this situation and whether patients have returned successfully to peritoneal dialysis.

Key words
Hydrothorax, pleurodesis, diaphragmatic defect, peritoneal leak

Introduction
Hydrothorax is an uncommon but well-recognized complication of peritoneal dialysis (PD). First reported in 1967, hydrothorax occurs in up to 6% of adult continuous ambulatory peritoneal dialysis (CAPD) patients, according to a recent large U.S. study (1). Patients develop a transudative pleural effusion, usually on the right side. The condition can usually be readily differentiated from other causes of transudative pleural effusion such as congestive cardiac failure, hypoalbuminemia, or total body fluid overload.

Here, we describe a case of hydrothorax in a PD patient who underwent video-assisted thorascopic surgery (VATS) and talc pleurodesis to repair a defect in the diaphragm and preserve PD capability. We reviewed the literature for 2001 – 2005 to assess how others have handled this situation and whether patients have returned successfully to PD.

Case report
The patient was a 60-year-old African American female who had initiated PD without any difficulty 3 weeks before admission. At 1 week before admission, she transitioned to automated PD using 15 L of low-calcium 1.5% dextrose dialysate nightly. Several days before admission, she developed progressive dyspnea, which worsened acutely the night before admission. In the emergency department, she also complained of discomfort in her right back and flank with deep inspiration and of an occasional nonproductive cough, but she reported no fever or chills. A chest radiograph showed a large right pleural effusion, cardiomegaly, and a very small left pleural effusion versus atelectasis.

Her medications included amlodipine, epoetin alfa, sevelamer, ferrous sulfate, metoprolol, losartan, atorvastatin, doxercalciferol, and insulin. She was a lifetime nonsmoker.

Physical examination was notable for mild respiratory distress with prominent jugular veins; a blood pressure of 198/112 mmHg; oxygen saturation of 96% on 2-L/min oxygen by nasal cannula; a 2/6 systolic ejection murmur, which was old; absent breath sounds at the right lower lung field, with clear lung fields superiorly and on the left; and 2+ leg edema bilaterally, which was new. Her initial laboratory studies were unremarkable.

Her PD treatment was initially continued. A large-volume thoracentesis on hospital day 2 yielded 2 L of straw-colored fluid containing 11 leukocytes, 240 erythrocytes, glucose at 490 mg/dL (serum glucose was 192 mg/dL), lactate dehydrogenase at 20 mg/dL, and total protein at 0.2 g/dL. This analysis was considered to be consistent with a diaphragmatic leak of PD fluid, and the patient’s CAPD was discontinued. A postprocedure chest X-ray showed a decrease in the size of the right pleural effusion. Repeat chest X-ray showed a large right pleural effusion with right lower-lobe collapse and a clear left lung with a “tiny” effusion. On hospital day 5, a dialysis catheter was placed, and the patient began hemodialysis. The same day, she had a technetium scan that showed tracer accumulation in the chest, which was considered definitive evidence of peritoneal–pleural communication.

On hospital day 6, a right Pleurex catheter was placed, and her right pleural effusion was completely resolved.
drained. At that time, because of a lack of outpatient dialysis capacity that would necessitate a prolonged inpatient stay if this patient were to remain on hemodialysis, as well as a strong reluctance to do hemodialysis, a plan was set to rechallenge the patient with manual PD exchanges in an upright position in the hopes that she could tolerate the exchanges. On hospital day 8, manual PD was restarted using 1-L exchanges with the patient sitting up at least at a 45-degree angle. A chest X-ray the following day showed reaccumulation of the right pleural effusion. The PD was discontinued, and the patient was continued on hemodialysis.

On hospital day 14, she underwent chemical pleurodesis with doxycycline via the right chest tube. She complained of significant pain in the right hemithorax immediately after instillation of the chemical agent; this pain lasted for approximately 1 week. She was continued on hemodialysis for the following week, then rechallenged with CAPD, but reaccumulation of a large right pleural effusion was seen on repeat chest X-ray. The PD was again discontinued, the effusion was drained, and hemodialysis was continued.

On hospital day 27, the patient was taken to the operating room, where she underwent thoracostomy and fluoroscopic examination of the entire surface of the diaphragm, which showed one focal area with a diaphragmatic bleb (Figure 1). The defect was repaired directly via thoracotomy and suturing, and then 4 g aerosolized talc was sprayed into the chest cavity.

The patient was maintained on hemodialysis for the next 4 weeks. On hospital day 54, she was rechallenged with CAPD using 1-L exchanges, which were increased to 2-L exchanges after a chest X-ray showed no reaccumulation of the effusion the next day. Follow-up chest X-rays showed no reaccumulation of the effusion. Follow-up outpatient appointments over the next 9 months showed no sign of recurrence of the effusion.

Discussion

Epidemiology

Pleuroperitoneal leaks commonly occur within the first few months after initiation of PD. A female predominance of 61% is seen. Right-sided hydrothorax is more common than left-sided (2).

Pathogenesis

Szeto and Chow (2) undertook a review of the pathogenesis of hydrothorax in 2004, and more recently VATS has been providing growing evidence that pleuroperitoneal defects result in hydrothorax (3–5). Whether these defects are congenital or acquired is not clear. Congenital defect might explain the preponderance of right-sided hydrothorax, because left-sided defects are covered by the heart and pericardium, thereby protecting against the leak.

Diagnosis

Leblanc et al. reviewed the diagnostic investigation of pleuroperitoneal leaks (6). Thoracentesis is the traditional means of hydrothorax assessment. In a small study, Chow et al. achieved 100% sensitivity and specificity for detecting pleuroperitoneal communication if the glucose gradient between the pleural fluid and serum glucose was more than 50 mg/dL (7).

Imaging approaches consist of adding a dye or isotope to the peritoneal fluid and then checking for migration of the marked fluid into the thoracic cavity (8).
Recently, a method using CO₂ injected through the peritoneal catheter, with visualization of the diaphragm under water, has been described to identify otherwise hidden leaks, which can then be directly repaired (9).

**Treatment**

Traditionally, the preferred management of hydrothorax complicating PD has been interruption of PD for a period of 2 – 6 weeks (10). The rationale is to allow the parietal and visceral pleural surfaces to fuse spontaneously and to avoid potentially unnecessary surgical intervention. However, this approach is successful in only 52% of cases (Table I).

Instillation of sclerosing agents into the pleural cavity (pleurodesis) leads to an inflammatory reaction and pleural fibrosis that may obliterate the pleuroperitoneal communication. Unfortunately, the success rate of this technique is only 47%, and it can be associated with significant patient discomfort (Table I). Failure of chemical pleurodesis was thought to be attributable to large pleuroperitoneal defects that are not amenable to closure by pleurodesis.

Since the early 1990s, the advantages of VATS over conventional thoracotomy have been increasingly recognized in a variety of surgical settings. This minimally invasive procedure permits excellent visualization of the entire parietal pleura and diaphragmatic surface. Several studies have documented the efficacy of this approach in the management of pleuroperitoneal leaks in PD, with an overall success rate in the literature of 84% (Table I).

**Conclusions**

Hydrothorax in the PD setting occurs infrequently, but may be more common than previously recognized. There is no recognized standard of care for its management. Simple dialysis interruption or chemical pleurodesis by chest tube are low-risk and low-cost options, but are effective in only about half of cases. The minimally invasive VATS procedure allows for visualization of the diaphragmatic surface to evaluate for leaks that could result in failure of a more conservative method. The VATS technique also permits the defect to be repaired and a sclerosing agent to be evenly distributed over the diaphragm.

Our case demonstrates a combination of VATS repair of the diaphragm with talc pleurodesis and subsequent successful return to PD. To date, no reports of a combination of repair and talc pleurodesis have appeared in the literature.

A literature review indicated that the usual initial strategy appears to be PD interruption for a period of 4 – 6 weeks. If the hydrothorax then recurs, the most effective approach appears to be VATS with direct visual evaluation for diaphragmatic leaks, possibly including use of the air leakage method, and direct repair of identified defects or talc pleurodesis of the diaphragmatic surface if no defects are identified.

**TABLE I** Hydrothorax complicating peritoneal dialysis (PD) in adults

<table>
<thead>
<tr>
<th>Ref.</th>
<th align="right">Cases</th>
<th>Study period</th>
<th>Mean age (years)</th>
<th>Median onset (months after PD start)</th>
<th>Sex [n (%)]</th>
<th>PD interruption alone</th>
<th>Success rates [n (%)]</th>
<th>Chemical pleurodesis via chest drain</th>
<th>Thoracotomy</th>
<th>Thoracoscopy</th>
</tr>
</thead>
<tbody>
<tr>
<td>11</td>
<td align="right">5 (5.1)</td>
<td>1978–1983</td>
<td>58</td>
<td>0.3</td>
<td>5 (100)</td>
<td>—</td>
<td>1/2 (50)</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>12</td>
<td align="right">50 (1.6)</td>
<td>1980–1988</td>
<td>49</td>
<td>1</td>
<td>23 (46)</td>
<td>19/35 (54)</td>
<td>8/15 (53)</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>13</td>
<td align="right">4 (3.3)</td>
<td>Before 1989</td>
<td>58</td>
<td>1</td>
<td>4 (100)</td>
<td>—</td>
<td>2/2 (100)</td>
<td>2/2 (100)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>14</td>
<td align="right">5</td>
<td>Before 1988</td>
<td>53</td>
<td>3</td>
<td>4 (80)</td>
<td>1/1 (100)</td>
<td>0/2 (0)</td>
<td>2/2 (100)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>15</td>
<td align="right">5 (1.9)</td>
<td>1986–1992</td>
<td>50</td>
<td>2</td>
<td>5 (100)</td>
<td>—</td>
<td>1/5 (20)</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>16</td>
<td align="right">4 (3.1)</td>
<td>1986–1997</td>
<td>Not reported</td>
<td>2</td>
<td>50 (%)</td>
<td>—</td>
<td>0/2 (0)</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>7, 17</td>
<td align="right">9 (1.0)</td>
<td>1986–2001</td>
<td>46</td>
<td>2</td>
<td>8 (88)</td>
<td>1/1 (100)</td>
<td>4/5 (80)</td>
<td>1/2 (50)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>18</td>
<td align="right">5</td>
<td>1991–1999</td>
<td>38</td>
<td>3</td>
<td>3 (60)</td>
<td>1/1 (100)</td>
<td>1/3 (33)</td>
<td>1/1 (100)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>5, 19</td>
<td align="right">8 (2.0)</td>
<td>1994–1998</td>
<td>46</td>
<td>2</td>
<td>4 (50)</td>
<td>1/2 (50)</td>
<td>0/2 (0)</td>
<td>—</td>
<td>5/6 (83)</td>
<td>—</td>
</tr>
<tr>
<td>8</td>
<td align="right">9 (1.9)</td>
<td>1998–2002</td>
<td>53</td>
<td>5</td>
<td>6 (67)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>9/9 (100)</td>
<td>—</td>
</tr>
<tr>
<td>20</td>
<td align="right">1</td>
<td>2004</td>
<td>53</td>
<td>Not reported</td>
<td>0 (0)</td>
<td>0/1 (100)</td>
<td>0/1 (100)</td>
<td>—</td>
<td>0/1 (0)</td>
<td>—</td>
</tr>
<tr>
<td>Current case</td>
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<td>2005</td>
<td>60</td>
<td>0.7</td>
<td>1 (100)</td>
<td>0/1 (100)</td>
<td>24/46 (52)</td>
<td>16/34 (47)</td>
<td>5/5 (100)</td>
<td>16/19 (84)</td>
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<tr>
<td>Totals</td>
<td align="right">106</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>65 (61)</td>
<td>—</td>
<td>24/46 (52)</td>
<td>16/34 (47)</td>
<td>5/5 (100)</td>
<td>16/19 (84)</td>
</tr>
</tbody>
</table>
Our case also illustrates that talc pleurodesis may be performed even if a diaphragmatic defect is identified; the pleurodesis will seal any other potential defects that are not identified. Given the possibility that some defects cannot be seen, the lack of side effects or complications of talc pleurodesis by thoracoscopy makes this approach an option if the operator wishes to ensure that a procedure will be successful even when a diaphragmatic defect has been identified. If talc pleurodesis is used, we recommend a minimum of 4 weeks of PD rest after such a procedure to provide adequate time for sclerosis of the diaphragmatic defect. If repair of the visible defect alone is used, the patient may resume PD immediately.

References

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